Case Report

Management of Patients with Advanced Heart Failure According to Hemodynamic Parameters

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Introduction

Heart failure (HF) is characterized by progressive functional or structural worsening of the myocardium. Despite significant therapeutic advances that have improved survival and quality of life, HF still has high morbidity and mortality rates. Patients with HF may progress with refractory disease, whose gold standard treatment is heart transplantation (HT).[1,2]

The assessment of pulmonary hemodynamics is indicated for HT candidates. The presence of fixed pulmonary hypertension (PH) is a contraindication for HT because it promotes right ventricular (RV) dysfunction in the graft, reducing post-HT survival. In addition, the assessment of hemodynamic parameters helps in bedside therapeutic management, including in the setting of PH, favoring HT indication.[1,2] We report a successful case of optimal bedside therapy guided by invasive hemodynamic parameters.

Case report

A 58-year-old man with diabetes, stage D HF secondary to idiopathic dilated cardiomyopathy, and recurrent hospitalizations despite optimal drug therapy was referred for outpatient HT evaluation, with evidence of reversible PH after vasodilator testing (Table 1). The patient required hospitalization due to disease progression, pulmonary congestion, and peripheral hypoperfusion. Echocardiogram showed a left ventricular ejection fraction of 25% with diffuse hypokinesia, left ventricular (LV) diastolic and systolic diameters of 68 x 64 mm, moderate RV hypokinesia (S' wave 6 cm/s; TAPSE 26 mm), and a pulmonary artery systolic pressure (PASP) of 55 mm Hg, with severe tricuspid regurgitation. Due to clinical severity, the patient was started on intravenous inotropic, diuretic, and vasodilator support. The patient underwent a new hemodynamic assessment with the use of a pulmonary artery catheter (PAC) in an intensive care setting, with evidence of PH (Table 2). Since the patient's clinical condition was a contraindication for HT, the implant of a long-term ventricular assist device (VAD) was considered but not conducted due to social reasons. The patient was placed on the HT waiting list for heterotopic transplantation, and therapeutic support was optimized with a combination of milrinone, circulatory support via intra-aortic balloon pumping (IABP), and inhaled nitric oxide.

After 4 months on the waiting list, a new invasive evaluation with a PAC identified significant reduction in pulmonary pressures (Table 2), supporting orthotopic transplantation. The patient underwent an HT 5 months after hospitalization with no complications.

Discussion

The case reported here illustrates the impact of optimal therapy on the improvement of hemodynamic parameters, assessed by serial invasive evaluation with a PAC, in a patient with decompensated HF and cardiogenic shock. PH associated with heart disease, called postcapillary pulmonary hypertension, is characterized by elevation in filling pressures, mean pulmonary blood pressure (mPBP), and pulmonary capillary wedge pressure (PCWP) and constitutes a marker of disease progression in HF with reduced ejection fraction. PH is characterized by an mPBP > 20 mm Hg and a pulmonary vascular resistance (PVR) ≥ 3 Wood;[3,4] if PCWP > 15 mm Hg, PH is considered postcapillary. In this case, the increase in pulmonary artery pressure occurs by retrograde transmission of increased hydrostatic pressure from the left atrium into the pulmonary veins and capillaries.[5]

Elevated central venous pressure resistant to drug therapy may be considered a contraindication for HT. In patients with evidence of PH, testing with intravenous vasodilators should be performed to demonstrate whether PH is reversible. Continuous 24-hour to 48-hour monitoring with full therapy consisting of diuretics, inotropes, and intravenous and inhaled vasodilators should be encouraged in cases of irreversible PH.[6]

Long-term VADs are a therapeutic option in patients that cannot undergo an HT as they may promote LV decompression, reduction in filling pressures, and, consequently, reduction in pulmonary pressures.[7] VAD indication in Brazil in the setting of public health is limited due to socioeconomic conditions. In this case, heterotopic transplantation may be an option with limited results.

In heterotopic HT, the graft is connected to the native heart, which is maintained in the patient's rib cage, and acts as a biological LVAD. This procedure may be considered in patients with obesity or increased PVR. However, the feasibility of the procedure remains uncertain.

Improvement in LV systolic volume causes increased RV preload, which may result in poor RV performance and compliance. Therefore, the presence of previous RV dysfunction

Keywords

Management; Advanced Heart Failure; Hemodynamics

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Table 1 – Right heart catheterization before hospitalization

	Before vasodilator testing	After vasodilator testing
CO	2.8 L/min	2.8 L/min
CVP	12 mm Hg	5 mm Hg
PBP	74 x 30 mm Hg	26 x 11 mm Hg
mPBP	44 mm Hg	17 mm Hg
PCWP	25 mm Hg	5 mm Hg
TPG	19	12
DPG	5 mm Hg	6 mm Hg
PVR	6.7 Wood	4.2 Wood
PAPP	44 mm Hg	15 mm Hg
PAPi	3.6	3.0

CO: cardiac output; CVP: central venous pressure; DPG: diastolic pulmonary gradient; mPBP: mean pulmonary blood pressure; PAPi: pulmonary artery pulsatility index; PAPP: pulmonary arterial pulse pressure; PBP: pulmonary blood pressure – diastolic and systolic; PCWP: pulmonary capillary wedge pressure; PVR: pulmonary vascular resistance; TPG: transpulmonary pressure gradient.

Table 2 – Progression of hemodynamic parameters during hospitalization and after 4 months of optimal guided therapy

	During hospitalization	After 4 months
CO	5.4 L/min	6.6 L/min
CVP	5 mm Hg	21 mm Hg
PBP	50 x 22 mm Hg	58 x 33 mm Hg
mPBP	30 mm Hg	41 mm Hg
PCWP	9 mm Hg	32 mm Hg
TPG	21	9
DPG	13 mm Hg	1 mm Hg
PVR	3.8 Wood	1.3 Wood
PAPP	28 mm Hg	25 mm Hg
PAPi	5.6	1.1

CO: cardiac output; CVP: central venous pressure; DPG: diastolic pulmonary gradient; mPBP: mean pulmonary blood pressure; PAPi: pulmonary artery pulsatility index; PAPP: pulmonary arterial pulse pressure; PBP: pulmonary blood pressure – diastolic and systolic; PCWP: pulmonary capillary wedge pressure; PVR: pulmonary vascular resistance; TPG: transpulmonary pressure gradient.

is a contraindication for both VAD implantation and heterotopic HT, which were not good options for our patient.[8]

Optimal therapy with parenteral and inhaled vasodilators promoted PASP and PCWP reduction, resulting in a decreased transpulmonary pressure gradient and increased cardiac output by reductions in RV afterload, LV preload, and, consequently, PVR. Hypervolemia reduction, on the other hand, promoted reduction in pulmonary pressures.[1] Dobutamine acts on the beta-1 adrenergic receptor increasing calcium influx and resulting in myocardial contractility. Milrinone is a phosphodiesterase-3 inhibitor that is involved in cyclic guanosine monophosphate degradation, leading to an increase in calcium influx and inotropism.[9] Due to phosphodiesterase inhibition, pulmonary vasodilation with a consequent reduction in PH and optimal RV afterload were observed.[7] The mechanism of action of IABP is aortic counterpulsation, aortic root diastolic pressure augmentation, afterload reduction, and, consequently, CO increase.[7,9]

The use of mechanical circulatory support should be considered in patients with a potentially reversible disease and pharmacologically irreversible PH. According to the International Society for Heart and Lung Transplantation, the use of mechanical circulatory support in the management of patients with HP is a class IIB recommendation.[6.]

The Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) reported adverse events associated with the use of PACs, such as arrhythmias, sepsis, pulmonary artery perforation or rupture, and even death. Therefore, the risks of PACs outweigh the benefits, leading several guidelines to not indicate pulmonary artery catheterization.[10] However, if used with caution in combination with risk minimization techniques, PACs could help optimize patient support, as occurred in the case reported here.

Conclusion

The advanced stages of HF are challenging from a therapeutic perspective, especially when deciding on the optimal destination therapy. PH is a marker of advanced HF, and the use of invasive monitoring may be useful to optimize bedside therapy and to adjust hemodynamic parameters that allow for HT.

Author Contributions

Conception and design of the research, Acquisition of data, Analysis and interpretation of the data, Statistical analysis, Writing of the manuscript and Critical revision of the manuscript for intellectual content: Aragão CAS, Costa DM, Ávila MS.

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This article does not contain any studies with human participants or animals performed by any of the authors.

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